repression of significant emotional events and no need to contain emotional conflict. Jancar's (1978) contribution to the discussion suggests that this is shared by the severely mentally handicapped, not many of whom would have been taking phenothiazines, a possible causal agent put forward by Schiff (1979).

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ANOTHER CASE OF KORO IN A BRITON

DEAR SIR,

Over the last fifteen years, at least three cases of koro-like symptomatology have been described in Westerners (Barrett, 1978; Edwards, 1970; Yap, 1965). I would like to report another recent case of koro-like symptoms occurring in an Englishman.

The patient is a young man of 24, who has never been out of Europe. He has one aunt who was repeatedly admitted to hospital suffering from a depressive illness, and both his parents are described as being of nervous disposition. Apart from enuresis up until the age of eight, the first hint of abnormality in his personal history was at the age of twelve, when he began cross-dressing in his sister's clothes and masturbating. There were no homosexual fantasies. He continued to do this once or twice a week until the age of eighteen. Meanwhile there was a very insidious onset, from about the age of fourteen, of irrational fears, obsessive-compulsive symptoms, hypochondriacal complaints, labile mood, outbursts of violence, heavy drinking, and recurrence of nocturnal enuresis. There was no history of drug abuse.

At the age of eighteen he attempted sexual intercourse on three separate occasions, and was impotent. He became extremely upset about this, began drinking heavily, and developed numerous fears about his health, including a worry that there might be something wrong with his penis. In 1977, at the age of 21, he was admitted to a psychiatric hospital suffering from severe anxiety associated with obsessional thoughts and rituals.

After discharge he was walking down a street one afternoon when he suddenly felt his penis shrinking to 'about half an inch in length'. He became very frightened, returned home, examined himself, and claimed that he could see and feel that his penis was disappearing into him. He feared that it might disappear altogether, and that he would then die or have to kill himself. Since then he claims to have this 'shrinking' sensation all the time, with more acute episodes occurring almost every day, during which he feels his penis becoming even smaller. At these times he becomes extremely agitated, panicky, and distressed. He believes that he may have a very rare physical illness, although he has never heard of koro himself, or met anyone with similar symptoms.

Later, he became very depressed and withdrawn, as he felt that the shrinkage of his penis must be immediately apparent to everyone. In early 1969 he began hearing a single male voice which called him unpleasant names, ordered him to carry out various rituals, and threatened him with his mother's death. He also developed ideas of being controlled by outside agencies.

In July 1979 he was admitted to hospital. Physical examination showed nothing abnormal. He was withdrawn from alcohol, and treated with psychotherapeutic interviews and a variety of drugs including amitriptyline, combined amitriptyline and phenelzine, pimozide, chlorpromazine and haloperidol. However there was little improvement in his overall state, and two years later, his acute episodes of koro-like symptoms are as frequent and distressing as ever.

This patient is similar to the cases described by Yap (1965a and 1965b), including the case he described in a Briton, in many ways: the young adult age, being an only son with over-dependence on his mother, the story of unusual sexual conflict and maladjustment, and the associated symptoms of hypochondriasis, depression and heightened self-observation of the genitals before the onset of the classical feeling of the penis shrinking.

Diagnostically he remains a puzzle, exhibiting obsessional traits, depression, auditory hallucinations, and vague feelings of being controlled. Obsessional traits and schizophrenia-like symptoms occurred in Edwards' (1970) case in America, and Yap reported six cases against a schizophrenic background. The features of the koro syndrome appear to be less culture-bound than was originally thought.

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L-TRYPTOPHAN IN MATERNITY BLUES

DEAR SIR,

We were most interested to read Dr Harris' report (*Journal*, September 1980, **137**, 233–35) of a double-blind trial of L-tryptophan in the puerperium. The failure of exogenous L-tryptophan to affect the incidence of severity of maternity blues is probably not surprising. In a recent study (*Journal*, May 1980, **136**, 498) we showed that whether or not the blues is associated with lowered free tryptophan is strongly affected by seasonal factors. In addition, we showed that, in our subjects, failure of total tryptophan to rise after parturition was a significant indicator not only of the blues but of complaints of depression in the ensuing six months.

It is difficult to see how such a brief disturbance of tryptophan at parturition could bear a causal relationship to outcome at six months and accordingly we suggested that it may indicate an occult disturbance in tryptophan handling, perhaps related to a more generalized membrane transport disorder, which may be a predisposing factor for the development of depression. Thus we envisaged that disturbances in tryptophan dynamics during the puerperium could be biological markers of susceptibility to depression rather than primary causative factors. Dr Harris' findings support this view and in this sense disturbances in total or free tryptophan at parturition may indeed be epiphenomena as he suggests, not of the blues, but of a more fundamental disturbance which does bear a causal relationship to depressed mood.

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THE ART OF MEASURING SEROTONIN UPTAKE IN PLATELETS

DEAR SIR,

Arora and Meltzer (*Journal*, October 1980, **137**, 396–99) criticize the paper by Coppen *et al* (*Journal*, March 1980, **136**, 235–38), mainly on methodological grounds. Their main point seems to be that when 5-HT uptake is determined at concentrations greater than 1 μM of 5-HT, the effects of passive diffusion cannot be entirely corrected. According to my studies (and those of others) this is not correct—provided one uses an adequate 'blind value' to be subtracted from the measured uptake values (Lingjaerde, 1977). The easiest way to obtain this blind value is that used by Coppen *et al:* to add varying concentrations of 5-HT to samples kept in the cold. When this blind value is used, I find that the initial uptake rate at 37°C shows all the characteristics of saturable, active uptake, at least up to 8 μM of 5-HT. Thus, it is completely blocked by low concentrations of antidepressants like clomipramine, and by omitting sodium or chloride from the incubation medium.

It may be possible to obtain the same data also by extrapolating back from the linear part of the uptake vs. concentration curve, as recommended by Stahl and Meltzer (1978), Tuomisto and Tukiainen (1976) and Tuomisto *et al* (1979). However, I find this method less satisfactory, because of the uncertainty in assessment of the 'linear part' of the (hyperbolic) uptake curve.

Like Arora and Meltzer, I was surprised by Coppen *et al's* finding of reduced 5-HT uptake in plasma after adding lithium carbonate. In my own *in vitro* studies with lithium (added as chloride) I have never seen an inhibitory effect of lithium, even in high concentrations. Neither have I seen a stimulatory effect, except in the absence of K^+; lithium thus seems to have a 'potassium-like' effect on 5-HT uptake (Lingjaerde, 1977). However, 5-HT uptake decreases very rapidly with increasing pH (Lingjaerde, 1977). Could it be that the inhibitory effect of lithium carbonate found by Coppen *et al* is due simply to increased pH?

Indeed, the measurement of 5-HT uptake in platelets is blessed with many pitfalls!

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